"Mirror-Image" Bilateral Giants: Intracavernous Carotid Artery Aneurysms

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Summary

The literature on the incidence of "mirror image" bilateral giant intracavernous aneurysms, their symptoms and their association with other entities is reviewed, with a brief comment on their evolution and treatment.

A case of "mirror image" bilateral giant intracavernous aneurysms in a 76-year-old man who presented a sudden diplopia with pupillary sparing is reported. A CT scan showed parasellar images and dolichomega circle of Willis arteries that enhanced with endovenous contrast. MRI and angiography disclose bilateral aneurysms in detail, associated with an anomalous origin of the left common carotid artery and bilateral renal artery stenosis.

Introduction

One to five percent of intracranial aneurysms are located in the intracavernous segment of the internal carotid artery where giant aneurysms are also frequently found [1,2,3].

Bilateral aneurysm of both intracavernous carotid arteries has been regarded as a rare finding [4,5,6]: Eguchi et Al. reported 28 cases in their paper in 1982 [7], Linskey et Al.[1] observed it in 21% of their cases of intracavernous carotid aneurysms which would suggest that their bilateralism is not so rare as had been thought.

The incidence of "mirror-image" bilateral giant intracavernous carotid artery aneurysms has not been defined to date. In many reported cases, they were not symmetrical in location or size. An association of such aneurysms with bilateral stenosis of renal arteries [8,9] and anatomical variants in the origin of the aortic vessels has been even less frequent.

We also report a case of "mirror-image" bilateral giant intracavernous carotid artery aneurysms associated with an anatomical variation consisting in the common origin of the left internal carotid with the bracheocephalic arterial trunk, and stenosis of both renal arteries.

Case Report

A 76-year-old man presented permanent vertical diplopia without associated headache five days prior to consultation. He had suffered an acute myocardial infarct nine years earlier.

On ophthalmoscopic examination there was grade II angiosclerosis, anisocoria due to left mild pupillary dilation with bilaterally preserved photomotor reflexes, paresia in left eye downward movements, and mild ipsilateral palpebral ptosis.

A contrast enhanced CT scan revealed an aneurysmatic enlargement of both carotid syphons (figures 1 and 2). A magnetic resonance scan showed their patency, with the hypophysis laterally displaced and reduced to a strip that homogeneously reinforced after administration of gadolinium (figure 3). The basilar artery was elongated, with a mild increase in

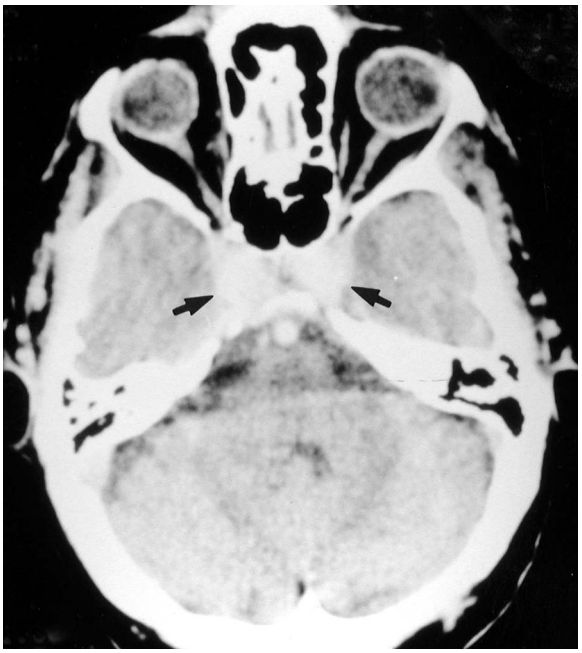

Figure 1 Axial contrast enhanced CT scan shows bilateral parasellar hyperdense lesions (arrows).

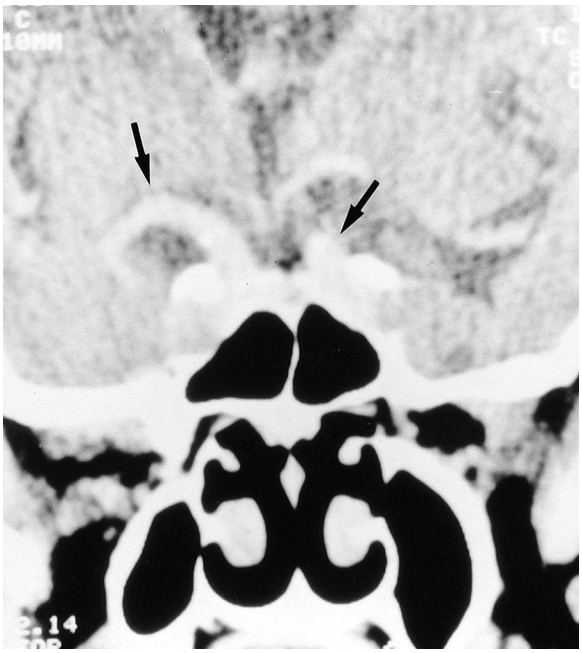

Figure 2 Coronal contrast enhanced CT scan through the sellar region shows bilateral intracavernous aneurysms and ectasic carotid arteries (arrows).

caliber that caused elevation and forward shifting of the mamillopontine region, distorting the third ventricle (figure 4).

In proton density and T2 images, there were hypertensive focal areas in the white matter of both brain hemispheres and the left cerebellar hemisphere. A MRI scan angiography showed mirror-image bilateral giant intracavernous aneurysms, with tumor-like growth at the sella turcica (figure 5).

Seven days after the first examination, the patient presented left retroocular headache and increased diplopia. An aortography showed a marked flexuosity of the aortic arc and primitive carotid arteries, the left one arising from a common trunk with the right one (figure 6). The bifurcations of both carotids did not present stenosis and giant fusiform aneurysmal dilatations lay on the intracavernous carotid segments (figure 7).

Supraclinoid segments of both carotids, and their branches, as well as the basilar and its branches were elongated but they did not present stenosis or aneurysms.

Further study with thoracic and abdominal aortic angiography was performed to detect peripheral associations. The aorta and iliac arteries were dilated and elongated, with abundance of calcified atheromas. The right renal artery showed a mild stenosis (less than 50 percent) at the point of origin, while the left renal artery presented a moderate stenosis (6O to 70 percent) about one centimetre away from the ostium, associated with a mild reduction in size of the ipsilateral renal parenchyma (figure 8). The superior mesenteric artery was appropriately opaque and the celiac trunk showed a flexuous course.

Prolactin and serum somatotropin values were 5.8 ng/ml and 0.46 ng/ml respectively (normal values: 3-13 ng/ml and up to 5.0 respectively in men).

After ten days of hospitalization, the patient's condition improved; he was free from diplopia and headaches. Later on he suffered two transient episodes of diplopia and paresthesia of the first and second branches of the left fifth nerve, which lasted between seven and 20 days, and spontaneously subsided.

The case was assessed jointly with the Service of Neurosurgery. It was agreed that no surgical or endovascular treatment was required because the symptoms had spontaneously subsided; the patient's age and clinical condition were also taken into account.

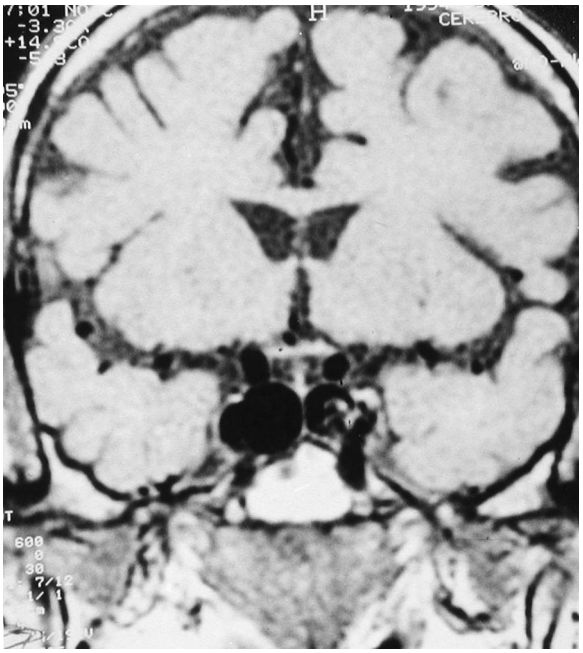

Figure 3 Coronal TI W MR scan shows bilateral aneurysms and thinned pituitary gland in the midline.

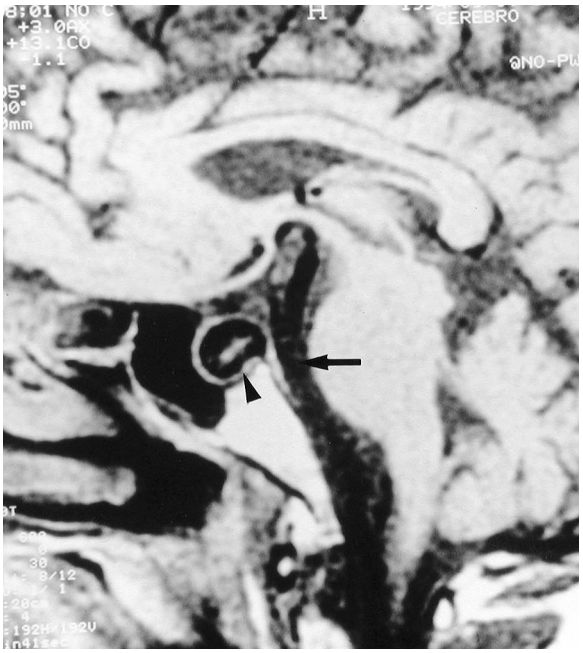

Figure 4 Sagittal TI W MR scan shows enlarged basilar artery (arrow) and aneurysm occupying the sellar fosa (arrowhead).

Discussion

Aneurysms of the intracavernous portion of the internal carotid account for 14% of all carotid aneurysms[1,10], and 3-5% of all intracranial aneurysms [1,11,12,13,14,15]. Multiple intracranial aneurysms, a group to which bilateral carotid aneurysms belong, have been observed in 15 to 23% of patients, of which 74 percent were women [1,10,16].

References on the incidence of bilateral intracavernous carotid aneurysms are scarce in the literature; they are mostly reports of isolated cases.

Eguchi et Al [7] published a case review in 1982 in which they reported only 28 cases. In 1990, Linskey et Al made an extensive review of a series of aneurysms and found 14 cases more, to which they added seven cases of their own. In this report, a series of 37 patients with intracavernous internal carotid aneurysms is presented of which 41% had multiple aneurysms, and 21% had bilateral intracavernous aneurysms [1].

In a study on the anatomical, clinical, and imaging features of aneurysms of the ophthalmic segment of the carotid artery (originating at the medial or anteromedial wall of the

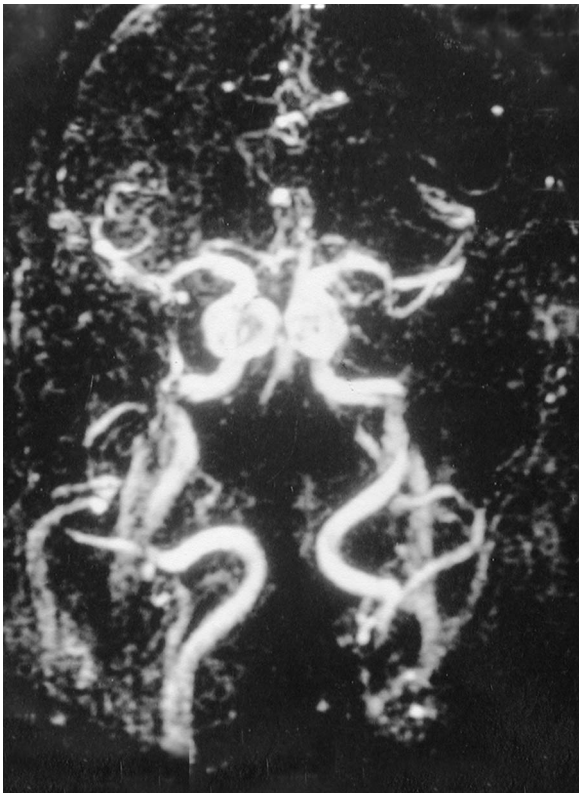

Figure 5 MR angiogram in AP view shows bilateral enlargement of both intracavernous carotid segments.

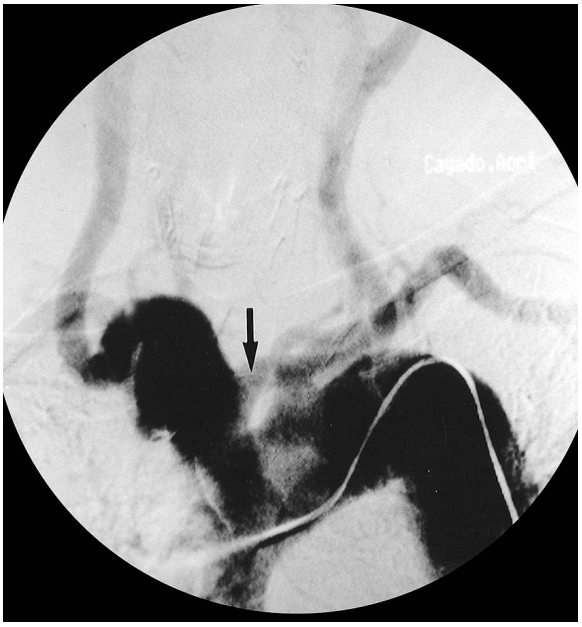

Figure 6 Aortic angiogram shows common origin of the innominate artery and left common carotid artery (arrow).

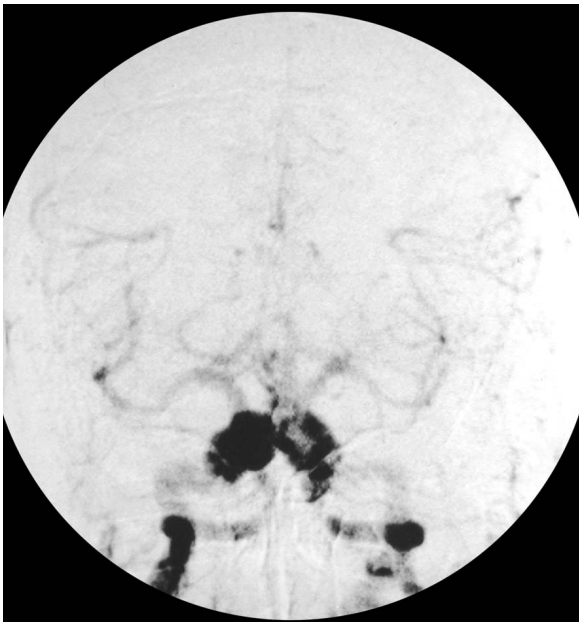

Figure 7 Intracranial angiogram AP view with both "mirror-image" intracavernous aneurysms.

internal carotid artery) in 80 patients, Day [17] found that 45% of those patients had at least one other intracranial aneurysm. In six out of 80 patients, there were bilateral aneurysms.

Aneurysms may be classified according to their size into: small aneurysms (<1 cm in diametre), large (1 to 2.5 cm), or giant (>2.5 cm). The majority of intracavernous carotid artery aneurysms are less than 2.5 cm (34% are small; 48% are large), while giant aneurysms (>2.5 cm in diameter) only represent 16% in Linskey's series, and vary between three and 39% in the literature [1,12,18,19]. Most giant aneurysms have been reported to arise from the anterior genu, others in the horizontal segment, and finally, from the posterior genu of the carotid artery[1].

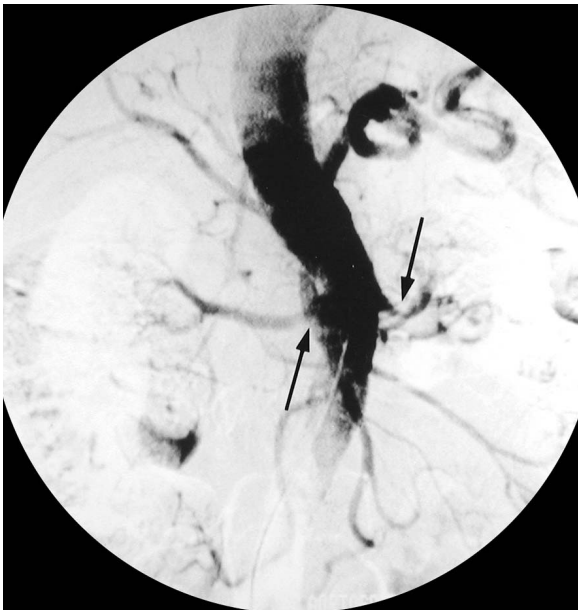

Figure 8 Abdominal aorta angiogram shows bilateral renal artery stenosis (arrows).

Intracavernous symptomatic aneurysms produce symptoms and signs of vascular or compressive origin. Vascular manifestations consist in subarachnoid hemorrhage, spontaneous cavernous carotid fistula, or generalised epistaxis due to rupture into the sphenoid sinus[3]. Subdural haematomas, embolic and distal ischemic phenomena, and thrombosis of the internal carotid artery are less frequent events [20]. Compressive phenomena are subject to the direction of the expansion of the aneurysmal sac. Anterior growth can produce superior orbital fissure syndrome [21]; posterolateral growth may erode into the petrous bone and cause hypacusia or hemorrhagic otitis; medial growth, by compressing the pituitary stalk, can result in disinhibition of prolactin secretion; finally, lat-

eral growth may lead to a cavernous sinus syndrome [1].

Pupillary behaviour is variable. Mydriasis due to lesions of parasympathetic fibres of the third nerve; myosis due to sympathetic alteration at the level of the pericarotid plexus, and medium sized, areflexic pupils have also been reported. Pupillary indemnity is frequently mentioned in this pathology in spite of third nerve participation [9,22-31]. A differential diagnosis is imperative in cases of myasthenia gravis with symmetrical or asymmetrical bilateral ophthalmoplegia, and neuropathy of the third nerve of diabetic origin in unilateral cases [28].

Among endocrine alterations, increase in prolactin has been reported which, added to ophthalmoplegia and headaches, demands a differential diagnosis with prolactinoma [28,32,33], although some associations of intracavernous aneurysms with somatotropic adenomas have also been reported [33-38].

No association of intracavernous aneurysms with bilateral renal obstruction has been found in the literature, except for one patient with fibromuscular dysplasia reported by Hirsch et Al. in 1975 [8,29]. In this case, aortic angiographic imaging compatible with atherosclerosis, added to the cardiac precedent and the absence of characteristic arterial lesions, does not allow for a diagnosis of fibromuscular dysplasia, generally associated with non-giant saccular aneurysms [29,39], or pseudoaneurysms due to dissection of the arterial wall.

Prognosis of giant aneurysms in general is that they are tolerable, as the aneurysmal sac growth is slow, and possibilities of rupture - in cases of strictly intracavernous aneurysms - are scarce. Only 1.4% develops subarachnoid haemorrhage [1], a percentage that increases in cases of intracavernous aneurysms on the border with the ophthalmic segment [7].

Treatment of intracavernous aneurysms is indicated in cases of symptomatic aneurysm [19]. They can be surgically approached from the base of the skull [40], or through the transarterial route by selective embolization with preservation of the parent vessel, or by balloon trapping, depending on the aneurysm's characteristics and circle of Willis circulation [41]. In cases of asymptomatic aneurysms, invasive treatment should be avoided due to the benign nature of these aneurysms [19,42]

In our case, a conservative approach was adopted in view of the remission of symptoms. Should treatment have been indicated, it would have been applied to the symptomatic side only, avoiding surgical or endovascular trapping to prevent haemodynamic overcharge of the opposite carotid artery where an increase in blood flow might have induced an increase in the size of the corresponding aneurysmal sac, and risk of making it also symptomatic.

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